Chapter 39

DELAYED MANAGEMENT OF PARANASAL SINUS FRACTURES

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INTRODUCTION

In past conflicts, head and neck injuries accounted for between 16% and 21% of battle injuries.[1–3] A 6-year review from 2001 through 2007 noted an increased proportion of head and neck wounds in Iraq and Afghanistan compared with previous conflicts.[4] This has been attributed to improvements in body armor that have improved survivability while highlighting the difficulty of protecting the face without limiting sight, hearing, and communication. Trauma involving the paranasal sinuses typically occurs in conjunction with associated facial fractures, which are addressed elsewhere in this text. Injury to the sinuses themselves may lead to obstruction with subsequent infection, extension to surrounding structures, or formation of mucoceles/mucopyoceles. Violation of the sinuses at their interface with the skull base may result in cerebrospinal fluid (CSF) leak, which predisposes the patient to infectious complications such as meningitis, epidural abscess, and subdural abscess. Managing sinus trauma is relatively straightforward and will be described herein. However, the one sinus that has generated the most discussion in management is the frontal sinus. The vast majority of the literature available on frontal sinus trauma and management exists in the civilian literature, with obvious differences in injury patterns compared with military trauma. Most civilian injuries involving the paranasal sinuses and specifically the frontal sinus result from motor vehicle accidents and blunt force, with a minority from low-velocity projectiles. In contrast, most injuries in theater are a result of high-velocity projectiles and fragmentation injuries from improvised explosive devices. Treatment of early and late complications arising from sinus trauma is described, and an algorithm for approach to frontal sinus trauma at a Level 4 facility is presented.

ANATOMY

The paranasal sinuses are typically found as paired structures pneumatizing the facial skeleton and bordering several critical structures. The pneumatization of the sinuses and relatively thin sinus bones in many areas offer a survival advantage by providing a "crumple zone" to dissipate force from impacts, thereby sparing more critical structures (eg, the globe, optic nerve, and anterior and middle cranial vaults from injury). The maxillary sinuses form a portion of the orbital floor and contribute to the medial and lateral vertical buttresses of the midface. The pterygomaxillary space is immediately posterior to the maxillary sinus, with the nasal cavity making up the medial border. The posterior fontanelle is typically dehiscent with only mucosa separating the maxillary sinus from the nasal cavity proper. The maxillary sinus drains through its ostium lateral to the uncinate process in the lateral and inferior aspects of the ethmoid infundibulum.[5]

The ethmoid sinuses are a collection of aerated cells with variable pneumatization patterns. There are generally between 4 to 8 cells that border the medial orbital wall laterally and the skull base superiorly and medially. They are divided into anterior and posterior groups by the basal lamella of the middle turbinate, which turns from a vertical orientation anteriorly to a more horizontal orientation posteriorly and inferiorly. The anterior group drains into the osteomeatal complex that also comprises the drainage pathways of the frontal and maxillary sinuses. The posterior group drains into the sphenoethmoid recess and is bordered posteriorly by the sphenoid sinus. The variable aeration patterns of the ethmoid cells account, in part, for the heterogeneity in anatomy seen in patients. Some of these patterns are discussed because they may impact surgical management of the sinuses.

The agger nasi is the most anterior ethmoid air cell and is typically seen pneumatizing above and anterior to the middle turbinate's anteriorly based insertion. It is present in >90% of patients. The infraorbital or Haller cell is an ethmoid cell that is located along the floor of the orbit. This may impact the drainage of the osteomeatal complex by narrowing the ethmoid infundibulum. The sphenoethmoid or Onodi cell is seen pneumatizing above and lateral to the sphenoid sinus. This relationship is critical to identify preoperatively because the Onodi cell may expose the optic nerve, skull base, or internal carotid artery to injury during surgery. Several other ethmoid air cells have also been described with regard to the frontal sinus outflow. Bent et al[6] described cell types that may impact the frontal sinus drainage anteriorly:

- Type I is a single air cell above the agger nasi.
- Type II is a tier of cells above the agger nasi.
- Type III is a single cell above the agger nasi pneumatizing into the frontal sinus proper.
- Type IV is a cell that resides entirely within the frontal sinus.

Posteriorly based cells include

- the suprabullar cell that is a cell superior to the ethmoid bulla,
- a frontal bullar cell that is a suprabullar cell that extends into the frontal recess, and
- the supraorbital cell that pneumatizes posterior to the frontal outflow and over the orbit laterally.[7]

The frontal sinus develops from an extension of an anterior ethmoid cell that pneumatizes into the frontal recess. They are paired 85% of the time and aplastic in only 5% of patients.[8] The frontal sinus is shaped similar to an inverted pyramid with the narrowest portion, the frontal recess, resembling the shape of an hourglass. The borders of the frontal recess are the lamina papyracea laterally, the middle turbinate medially, the ethmoid bulla posteriorly, and the agger nasi or frontal beak anteriorly. The outflow of the frontal sinus may be narrowed by many of the previously noted ethmoid cells. The anterior table of the frontal bone is the densest bone in the facial skeleton, being approximately twice the thickness of the posterior table. The dural veins of Breschet are intimately related to the venous drainage of the frontal sinus, which can provide an intracranial route for extension of frontal sinusitis.

INITIAL EVALUATION

As with all trauma patients, priority must be given to Advanced Trauma Life Support protocols. The initial survey should identify life-threatening injuries with the secondary survey to identify less emergent injuries. The majority of patients with injuries involving the paranasal sinuses will be stable on transfer, and their fractures may be addressed in a delayed fashion. Attention must be paid to all diagnostic and treatment data provided on transfer to the Role 4 facility. This will typically include

- details of injuries sustained;
- signs and symptoms on initial presentation, including the Glasgow Coma Scale score;
- CT (computed tomography) scans; and
- records of any surgical procedures performed prior to evacuation.

Physical examination is performed, first noting any obvious deformities or soft-tissue injuries. Visual acuity should be evaluated, as well as extraocular movements when feasible. Forced ductions may be useful to verify gaze limitations. Pupillary response is assessed, with any deficits noted, which may indicate cranial nerve deficits or intracranial injury. Intraocular pressure is measured with a tonometer. The face should be palpated for step-offs and instability. Nasal fractures may be obscured by edema. Therefore, palpation should be performed and the septum visualized to exclude a septal hematoma. Fractures involving the nose and ethmoid bone should alert the clinician to the possibility of a naso-orbital-ethmoid (NOE) fracture. If an NOE fracture is suspected, one should measure the intercanthal distance. Normal intercanthal distance is around 30 mm, whereas telecanthus is defined as >45 mm. Occlusion may be altered if tooth-bearing bones of the maxilla and mandible are fractured. This may result in premature molar contact on one or both sides, deviation to one side, and an anterior open bite. Gentle rocking of the maxilla may reveal midface fracture, although impaction may also give the illusion of stability. Motor and sensory functions of the face should be carefully assessed and documented. Finally, the patient is examined for any evidence of otorrhea or rhinorrhea, which may indicate disruption of the skull base with resultant CSF leak.

DIAGNOSTIC STUDIES

The study of choice to evaluate the paranasal sinuses in the setting of trauma is a noncontrast CT scan. A fine-cut (<1.5-mm cut thickness) CT with triplanar reconstructions will elucidate the injuries sustained to the facial skeleton and paranasal sinuses in detail (Exhibit 39-1). Three-dimensional reformatting may also aid in planning for delayed repair. Fractures involving the floor of the orbit with herniation of orbital contents into the maxillary sinus are easily demonstrated and allow accurate measurements of the floor defect. Opacification of the maxillary sinus is commonly noted, and often represents edema and hemorrhage. This will typically clear if the maxillary ostium is unobstructed. Therefore, the ethmoid infundibulum should be examined for disruption. The lacrimal duct should be followed in its entirety and assessed for injury. Fractures of the lamina papyracea will be noted with potential herniation of the orbital contents into the ethmoid cavity. The skull base should be carefully examined to rule

comminution of the anterior and posterior tables should be noted. Particular attention should be paid to delineate whether a fracture line extends into the outflow of the frontal recess because this predisposes the patient to late complications, such as mucoceles or mucopyoceles. When the frontal sinus is opacified, the integrity of the dura adjacent to the fracture cannot be adequately assessed with a CT scan.

In the setting of facial and head trauma, rhinorrhea or otorrhea is suspicious for a CSF leak. If fluid can be collected, it may be sent for β_2-transferrin analysis.[10] The results of this test may take up to a week to receive; therefore, treatment should not be delayed pending the results of this test if the clinician is highly suspicious of CSF leak. The CT scan is carefully examined, especially along the ethmoid roof and planum sphenoidale. Should an occult CSF fistula be suspected, other studies may be pursued, such as radionuclide cisternography, CT myelography, or magnetic resonance phase-contrast cisternography. Any one of these studies may be used to assess for a CSF leak. However, they all require an actively leaking site.[9,11] Another very useful diagnostic study is the instillation of intrathecal fluorescein, which will be discussed in detail in a subsequent section.[12]

out fractures involving the cribriform plate and the ethmoid roof extending to the planum sphenoidale. Finally, the frontal sinuses need to be assessed. The anterior frontal sinus table is quite dense, requiring 800 to 1,600 pounds of force to cause a fracture.[9] If an anterior table fracture is identified, then there is a possibility of an associated intracranial injury. Any

MANAGEMENT

Due to their potential communication with the oral and nasal cavities, fractures of the paranasal sinuses should be considered contaminated. If surgical repair is deemed necessary, perioperative antibiotics can reduce the rate of wound infections.[13] An appropriate antibiotic choice should cover typical oral and nasal florae. Prophylactic antibiotics, however, have not been shown to reduce infection rates. Therefore, if observation is elected, then prophylactic antibiotics are not administered.[14,15] The more important factor affecting the rate of postoperative wound complications appears to be a delay in the repair of fractures.[15] Because of this finding, operative repair should not be delayed any longer than is necessary for stabilization and transfer of the patient.

Midface trauma is often accompanied by fractures extending into the paranasal sinuses. Early treatment both in theater and at the Role 4 facility is often aimed at restoring the contours of the facial skeleton. Delayed management of midface bony defects is addressed elsewhere in this text. In many instances, fractures involving the sinuses may be observed, reserving delayed management to those patients who present with late complications. As previously described, the maxillary sinus on initial radiographic examination may show opacification, which typically represents

blood products or orbital contents. After orbital floor repair, the maxillary sinus is expected to clear as long as the maxillary ostium remains patent. If the patient develops signs or symptoms of acute sinusitis, then antibiotic treatment may be initiated. Antibiotics should be culture-directed, whenever possible. If cultures are not available, the first-line choice of antibiotic is amoxicillin.[16] Most practitioners recommend a minimum duration of 10 days of antibiotics. However, there is no current consensus on the maximum duration for antibiotic treatment. Should the infection fail to resolve, imaging is usually indicated. If the ostium has been disrupted, the goal should be to restore mucociliary clearance. An endoscopic surgical antrostomy may be completed, taking care to incorporate any surgical opening with the natural ostium to avoid the phenomenon of recirculation, whereby mucus flows out of the natural ostium and drops back into the surgical antrostomy. It is important to be aware of any dehiscence of the lamina papyracea or orbital floor because an inadvertent orbital injury can occur when performing a maxillary antrostomy if these defects are not recognized preoperatively. Any foreign material or fragment felt to be a nidus for infection may be removed via a transnasal approach, with a maxillary trephination or Caldwell-Luc approach reserved as

adjuncts for difficult-to-reach locations in the maxillary sinus, such as the anterior-lateral wall and floor. If the sinus is felt to be nonfunctional, dependent drainage may be established with an inferior antrostomy or via a mega-antrostomy.[17] The endoscopic maxillary mega-antrostomy is completed by extending a large maxillary antrostomy to the posterior maxillary wall, anteriorly to the lacrimal bone and inferiorly through the posterior half of the inferior turbinate down to the nasal floor.

Late complications of fractures involving the ethmoid air cells include acute or chronic sinusitis, orbital or intracranial infections, and delayed development of mucoceles or mucopyoceles. Many cases of acute or chronic sinusitis may be managed with antibiotics (culture directed, if possible) with the addition of oral or intranasal steroids for patients suspected of having chronic mucosal disease.[18] Patients who continue to be symptomatic with evidence of sinusitis on CT scan are considered candidates for surgery. The majority of cases requiring ethmoidectomy are amenable to an endoscopic approach with mucosal-sparing techniques. The ethmoid cells are cleared, leaving the middle turbinate, skull base, and lamina papyracea skeletonized. When surgery is indicated, preoperative planning must include a meticulous review of the relevant radiological studies, which may reveal unrepaired fractures of the medial orbital wall and/or ethmoid roof. If there is an active CSF leak, it must be repaired, and this will be discussed in a subsequent section of this chapter. In the event of a dehiscent lamina papyracea, the surgeon may either decide to operate around the defect or repair the medial wall prior to performing sinus surgery. When operating in the setting of an unrepaired medial orbital wall, the surgeon should consider use of an intraoperative image guidance system and can intraoperatively ballot the orbit to endoscopically visualize areas of dehiscence. Mucoceles and mucopyoceles are also generally approached endoscopically, with simple marsupialization being sufficient in the majority of cases.

Complicated sinusitis may arise from extension of local disease, primarily from the ethmoid sinuses, to the orbit and has previously been categorized by Chandler et al.[19] Periorbital cellulitis denotes inflammation of the orbital tissues anterior to the orbital septum. There may be significant soft-tissue edema, but the extraocular movements are not impaired and visual acuity remains unchanged. This may be managed with oral antibiotics and close clinical follow-up. Orbital cellulitis is characterized by involvement of the postseptal orbit. In addition to soft-tissue edema, there will be significant pain with extraocular movement, gaze limitation, and a decrease in visual acuity. These

patients are managed with intravenous antibiotics, serial examinations, and contrast-enhanced CT scans. Ophthalmology should also be consulted to evaluate the patient. CT scans may reveal a subperiosteal abscess lateral to the lamina papyracea. Small abscesses may also be managed with intravenous antibiotics in a stable patient. However, surgical drainage may be necessary.[20] In the past, drainage was usually performed via external approaches, such as a Lynch incision; but, in recent years, endoscopic transnasal drainage has become more popular because it is equally as effective and avoids facial incisions.[21] The medial orbital wall adjacent to the abscess is endoscopically skeletonized, and a portion of the lamina is decompressed to allow drainage of the abscess. A Penrose drain may be left in place to prevent reaccumulation of purulent material in the orbit.

Further evolution of infection may result in an orbital abscess, which should be co-managed with an ophthalmologist. Because of the valveless veins in the face, infection can spread retrograde from the face and sinuses into the cavernous sinus and develop a thrombosis. A cavernous sinus thrombosis is characterized by proptosis, ophthalmoplegia, papilledema, superior orbital fissure syndrome, altered mental status, and often bilateral symptoms. Prior to the advent of antibiotics, this was nearly universally fatal. Modern treatment is primarily medical, with intravenous antibiotics and supportive care being paramount. Indications for anticoagulation remains controversial, but there is some evidence that anticoagulation may be beneficial early in the disease process.[22]

Injury to the lacrimal duct is often a sequelae of an NOE fracture and can be overlooked on initial evaluation because of the severity of associated presenting injuries and late presentation of symptoms. Delayed manifestation of lacrimal duct obstruction can present as dacryocystitis and will universally include epiphora.[23] Medial canthus disruption and lacerations involving the lacrimal canaliculi have typically been noted on initial survey and may have been previously repaired. If the canaliculi have not been repaired, they may be probed and repaired with a 6-0 monofilament suture and Crawford stents. However, the best results are obtained with repair within the first 24 hours after injury. If the canalicular system has been irreparably damaged, the surgeon can divert lacrimal drainage with a dacryocystorhinostomy and a Jones tube placement. Dacryocystorhinostomy is not typically recommended in the setting of acute dacryocystitis. Instead, the preferred treatment is antibiotics, incision and drainage, and elective surgery once the inflammation has subsided. An ophthalmologist is consulted to

confirm obstruction of the lacrimal duct using tests such as lacrimal probing with Jones I and II testing, nuclear medicine studies, or a dacryocystogram. Once obstruction of the lacrimal duct is confirmed, the dacryocystorhinostomy may be performed externally or endoscopically. Regardless of approach, the goal is marsupialization of the lacrimal sac into the nose. External approaches are well described in the ophthalmology literature. The otolaryngologist has a unique advantage of performing this procedure endoscopically. The nasal mucosa is elevated anterior and superior to the middle turbinate to expose the bone overlying the lacrimal sac. The soft lacrimal bone is then removed with through-cutting punches, and a diamond bur is used to remove the denser bone of the frontal process of the maxilla. Once the entire lacrimal sac is exposed, it is incised and the mucosal flaps everted into the nasal cavity. Crawford stents are brought through the superior and inferior canaliculi into the nose and then tied off. These may be left in place for weeks to months at the discretion of the surgeon. Late dacryocystorhinostomy may be expected to provide long-term patency in >90% of cases.[23]

Because of the central location of the sphenoid sinus, a significant amount of force is required to result in a fracture of this structure. Multiple major neurovascular structures travel in close proximity to the sphenoid sinus. The lateral border of the sinus abuts the cavernous sinus transmitting several cranial nerves (III, IV, V_1, V_2, and VI) and the internal carotid artery that continues toward the posterior-lateral border of the sinus. Superiorly and laterally, the optic nerve abuts the sphenoid sinus. Shearing injury to the carotid artery in its intracranial course may result in late complications, such as carotid-cavernous fistula or pseudoaneurysm. Therefore, any fracture extending to the carotid canal should be imaged with CT or magnetic resonance angiography to rule out damage to the carotid artery. Because the petrous segment of the carotid artery is very difficult to control or repair via external approaches, the endovascular repair is performed by a qualified interventional radiologist or endovascular neurosurgeon.

CSF leaks may be approached transcranially; however, this may result in significant edema due to the amount of retraction necessary to expose the roof of the sphenoid. Transnasal approaches have the advantage of exposing the CSF leak without having to retract the brain. Leaks located in the roof or posterior border may be accessed by a transnasal or transethmoid approach, whereas leaks located at the far lateral aspect may be approached via a transpterygoid approach through the posterior wall of the maxillary sinus. Nondisplaced

fractures in the absence of neurological signs or obvious CSF leak may be managed expectantly, with surgical drainage reestablished on an elective basis if clinically indicated.

Fractures involving the optic canal may reveal an obvious fragment impinging on the optic nerve. If the patient retains useful vision, pulsed intravenous steroids may be useful in salvaging function. Should further decrement in vision occur while on steroids, optic decompression may be undertaken in the stable patient. This may be approached via a transnasal route, with complete sphenoethmoidectomy performed for exposure. The thick bone over the optic tubercle is gently drilled with a diamond burr and the remainder of the bone overlying the optic sheath removed with fine curettes or picks.[24] Any bony fragments impinging on the nerve are removed. Alternatively, a transcranial or transorbital approach may be performed by the neurosurgeon or ophthalmologist.

CSF rhinorrhea may accompany fractures involving the posterior table of the frontal sinuses, the cribriform plate, the ethmoid roof, and the sphenoid. Large defects will usually be identified in theater and repaired in conjunction with the primary fracture repair by a multidisciplinary team to include a neurosurgeon, otolaryngologist, and oral-maxillofacial surgeon. Utilization of a bicoronal approach, in addition to providing excellent exposure of the upper third of the facial skeleton, also grants access to the pericranial flap that is the workhorse flap used to repair large dural defects. The reliable blood supply of this flap is provided primarily through deep branches of the supraorbital and supratrochlear arteries. Alternatively, an allograft dural substitute may be utilized if the pericranial flap is compromised or not available.

Posttraumatic CSF leaks seen at the Role 4 facility are usually because of untreated fractures or a persistent leak following initial repair. Leakage of CSF may not be noted initially in theater because of the edema-obstructing egress of fluid and subsequently presenting later as the edema subsides. CSF leaks associated with small defects or those not readily noted on imaging may be amenable to conservative measures. Conservative management consists of strict bedrest and sinus precautions, with placement of a lumbar drain to reduce intracranial pressure at the discretion of the neurosurgeon (Exhibit 39-2). Many leaks treated in this fashion will resolve spontaneously within 5 to 7 days. If CSF rhinorrhea persists, repair is indicated; as studies show that the rate of meningitis rises substantially, the longer the surgery is delayed.[25] As previously discussed, fine-cut CT is the most helpful preoperative study in localizing the area of a suspected leak. CT cisternography and pledget studies may also be helpful. For

obvious or β$_2$-transferrin–confirmed leaks, intrathecal fluorescein can be advantageous for directly visualizing a leak for repair. Because this use is not currently US Food and Drug Administration (FDA) approved, the patient must be counseled appropriately about its FDA status and the risks of possible complications, including seizures and myelopathy. Overall, this method has proven quite safe when administered correctly by diluting 0.1 mL of 10% fluorescein with 10 mL of the patient's CSF and administered via the lumbar drain for >10 minutes.[26] This is done approximately 30 minutes prior to the expected time of visualization of the dehiscence. Once the leak is identified, the mucosa around the defect should be cleared by at least 5 mm. Multiple techniques and materials have been described for repair to include free mucosal autograft, fat graft, cartilage or bone grafts, dural substitute, acellular dermis, and vascularized flaps. Critical points of repair include

- watertight closure,
- avoidance of transplantation of the mucosa intracranially, and
- stabilization of the graft postoperatively.

The patient is placed on bedrest and sinus precautions, with any nonabsorbable packing removed in 5 to 7 days.

Much debate in the trauma literature has centered on the management of the frontal sinus following trauma. As previously described, between 800 to 1,600 pounds of force are required to fracture the anterior table.[9] Because of this, associated neurological injuries are common, and the patient should be assessed accordingly with consultation from neurosurgery as appropriate. Displaced fractures of the anterior table that do not involve the frontal outflow are repaired

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EXHIBIT 39-2

CONSERVATIVE MANAGEMENT OF CEREBROSPINAL FLUID LEAKS

Lumbar drain
Strict bedrest
Head of bed elevated 30°
Stool softeners
No straining
No nose blowing
Cough/sneeze with mouth open
Cough suppressants
Antiemetics
Blood pressure management

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for cosmetic purposes only. Observation may be acceptable for simple fractures, which are not expected to result in cosmetic deformity. A recent study suggested that fracture displacement <4 mm does not lead to notable contour irregularity.[27] Repair may be accomplished through a preexisting laceration, if available. Direct access via forehead rhytides, a gullwing incision, or unilateral brow incision may also be used. However, these approaches may limit exposure for fracture repair or be cosmetically unacceptable to the patient. If the frontal sinus fracture consists of two to three large fragments, a percutaneous method may be used for reduction, but sacrifices the ability to directly visualize and control the fragments.[28] The bicoronal approach provides the best overall exposure for comminuted fragments and fractures involving the frontal outflow or posterior table. Regardless of the approach to the anterior table, if there is any uncertainty about the status of the frontal recess or the posterior table, these areas should be directly and/or endoscopically visualized at the time of surgery.

If the frontal recess is disrupted by the fracture, the frontal sinus may not properly drain. Improper drainage can lead to chronic infection and obstruction, and subsequently result in mucoceles or mucopyoceles. Close proximity to the anterior cranial fossa and route of entry via the dural veins of Breschet put the patient at higher risk of intracranial complications if these conditions are not addressed. Involvement of the frontal recess is highly suspected with NOE fractures and, therefore, triplanar CT scans should be carefully reviewed to assess the outflow tract. As previously described, if the anterior table repair is planned and a frontal recess injury is suspected, it should be directly visualized. A topical decongestant may aid in determining patency. When the recess is determined to be irreparably damaged, frontal sinus obliteration should be considered.[29] Obliteration is best accomplished via a bicoronal approach, which allows the most comprehensive view of the frontal sinus. Once the flap is lifted, the borders of the anterior table are outlined with image guidance or transillumination. A previously described technique uses a precut template copied from a plain AP (anteroposterior) radiograph (the "six-foot Caldwell"). Bone cuts are beveled in toward the sinus to decrease risk of intracranial penetration. Once the sinus is exposed, the mucosa is meticulously removed and burred down with a diamond drill. If the posterior table is salvageable, it should be repaired at this time. The frontal recesses are stripped of mucosa and plugged with muscle or similar material. If a pericranial flap is being used, it may be laid along the floor of the frontal sinus. The frontal sinus is then filled with material to obliterate the space. The most commonly used material is fat. However, bone,

alloplastic material, and hydroxyapatite cement have been used. The authors prefer to use autograft material whenever possible. Proponents of obliteration feel that it is a long-term solution creating a safe barrier between the anterior cranial fossa and the nose. However, late complications are known to occur years following the procedure. It can be exceedingly difficult to remove all the mucus-producing elements in the sinus, with subsequent development of mucoceles and chronic infections. Some alloplastic materials have been associated with infections, necessitating complete removal. As endoscopic sinus surgery has advanced, some surgeons have elected to adopt a "watch-and-wait" policy, with endoscopic sinus surgery reserved for those who demonstrate persistent outflow obstruction or manifest complications. In addition, selected cases with failed obliterations may be managed endoscopically via reversal of the obliteration and reestablishment of endonasal drainage.[30] This may require extending techniques such as a modified endoscopic Lothrop procedure, which entails removal of the entire floor of both frontal sinuses, leaving one common drainage pathway (Figure 39-1).

Fractures involving the posterior table may be managed via observation, repair, or cranialization. The term cranialization refers to removal of the posterior table with the brain allowed to come forward, thus filling the space of the frontal sinus. Single fractures in the absence of a CSF leak may be observed if long-term follow-up and imaging are available. In the setting of severe disruption of the posterior table with a CSF leak, exploration and cranialization are generally preferred. A pericranial flap should be harvested when raising the bicoronal flap. Neurosurgery should be present to examine the anterior cranial fossa and perform any adjunctive procedures as needed. The bone of the entire posterior table and all of the sinus mucosa are removed, the frontal recesses are plugged, and then the pericranial flap is laid down. Generally, good, long-term results have been reported with this repair. Although cranialization is indicated in this setting, other institutions have reported managing comminuted posterior table fractures safely without having to perform cranialization.[31–33]
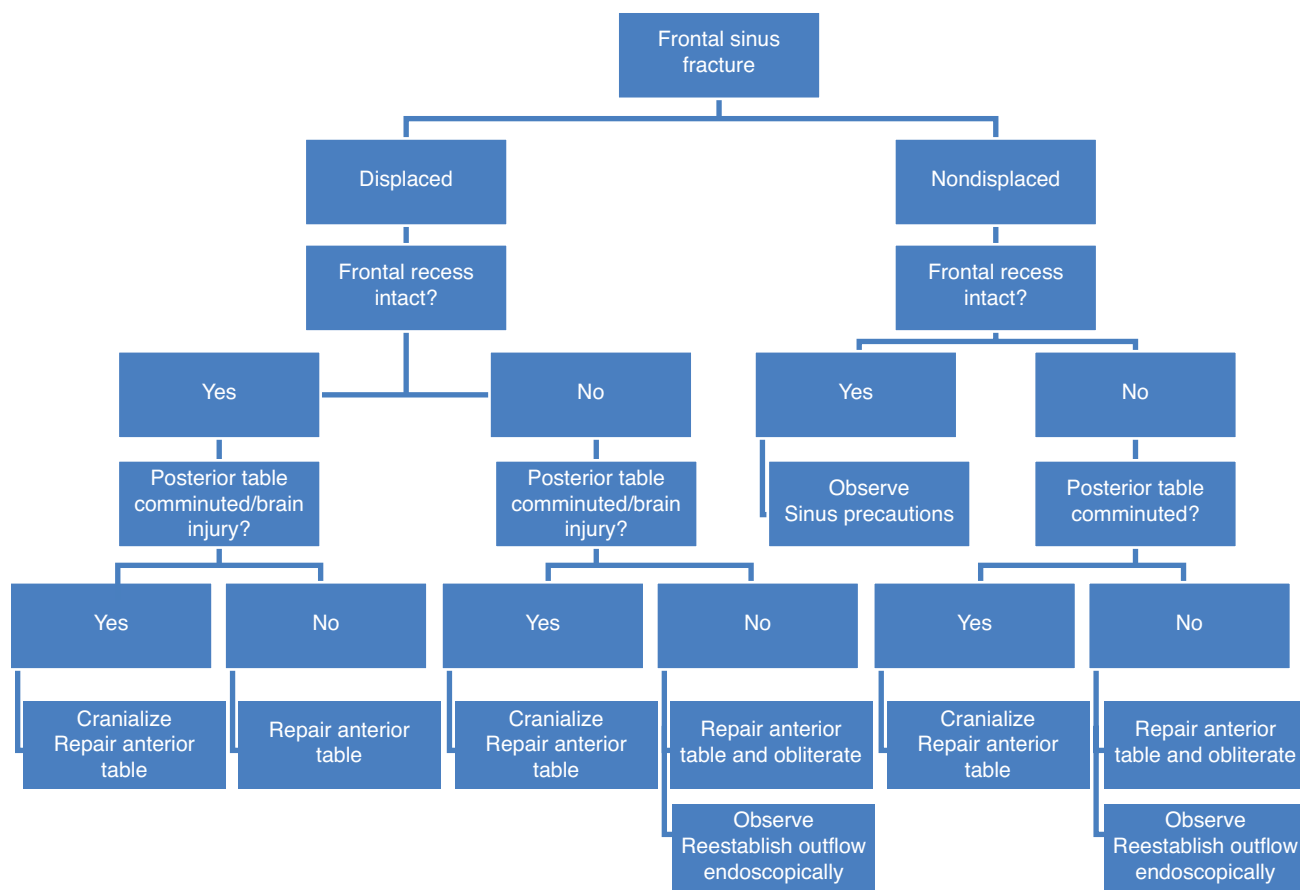

Figure 39-1. Management algorithm for frontal sinus fractures.

POSTOPERATIVE CARE

Postoperative care following surgical management of paranasal sinus fractures is tailored to the severity of the case. For simple cases of obstruction or mucocele, which have been relieved by endoscopic sinus surgery, no antibiotics are necessary. An exception is with the use of nonabsorbable packing, in which case prophylactic antibiotics are indicated. After uncomplicated sinus surgery, the patient may begin sinus irrigations with sterile saline the day following surgery, to be continued at the surgeon's discretion. All patients must follow strict sinus precautions to avoid insufflation of air into soft tissues through bony defects. Pain control is routinely accomplished through use of narcotics, nonsteroidal antiinflammatory drugs, or acetaminophen. Follow-up in the outpatient setting is performed at 1 week when any remaining absorbable packing is removed, and the operated cavities are examined endoscopically. Timing of subsequent follow-up visits is left to the surgeon.

Patients who have had CSF leak repair, frontal sinus obliteration, or cranialization require a higher level of care. After repair of CSF leaks, the patient may have a lumbar drain in place. This is managed in the intensive care unit. There is controversy regarding the utility of leaving a lumbar drain in place following repair. Some surgeons feel it is beneficial to keep intracranial pressure low by drawing off CSF, whereas others believe that the increased pressure improves the stability of the graft and repair. Initially, the patient should remain on strict bedrest with precautions similar to the nonoperative management of leaks. The patient is then slowly mobilized, with gradual return to normal activities as tolerated. In contrast to routine endoscopic sinus surgery, aggressive debridement following CSF leak repair is contraindicated. Following obliteration or cranialization, drains and pressure dressings are applied to prevent hematoma formation. The drains are removed 1 to 3 days after surgery. Pressure dressings are discontinued after 3 days. Transfer to a lower acuity of care is dependent on the patient's neurological status. Long-term follow-up is critical for these patients because of the risk of late mucocele infection. CT or magnetic resonance imaging should be performed if there is any evidence of recurrent infection or if mucocele is suspected.

SUMMARY

Definitive management of paranasal sinus fractures occurs at the Role 4 facility. Sinus fractures may result in simple obstruction or chronic infection, but may also be associated with significant orbital, neurological, or dental trauma. Simple injuries resulting in obstruction or infection may be followed with serial clinical examinations or imaging, with surgery reserved for failures of medical management. More complex maxillofacial and skull base injuries should be managed by a multidisciplinary team to include neurosurgery, otolaryngology, ophthalmology, and oral-maxillofacial surgery. The case example highlights the need for long-term follow-up in this patient population.

CASE PRESENTATION

Case Study 39-1

Presentation

A 36-year-old Army infantryman was evacuated from Afghanistan with a 6-month history of left visual loss greater than right visual loss. He also complained of intermittent right frontal headaches, sporadic right-sided clear rhinorrhea, and extremity weakness upon waking. He denied any head trauma in theater. Clinical examination revealed bitemporal hemianopsia and cranial nerve I, II, and III deficits. After CT scan was performed, he was transferred to Landstuhl Regional Medical Center (Landstuhl, Germany) for magnetic resonance imaging and then finally to Brooke Army Medical Center (Fort Sam Houston, Texas) for definitive care.

Diagnostic Studies

CT performed in theater showed a large, low-density area involving the right frontal lobe, as well as dehiscence of the roof of the posterior ethmoid extending into the sphenoid sinus with associated encephalocele (Figure 39-2). Magnetic resonance imaging with contrast showed T2 hyperintensity involving the right frontal lobe, felt to be a result of encephalomalacia versus potentially a cystic neoplasm (Figure 39-3). Further examination of the imaging demonstrated a previous fracture of the posterior table of the frontal sinus (Figure 39-4).
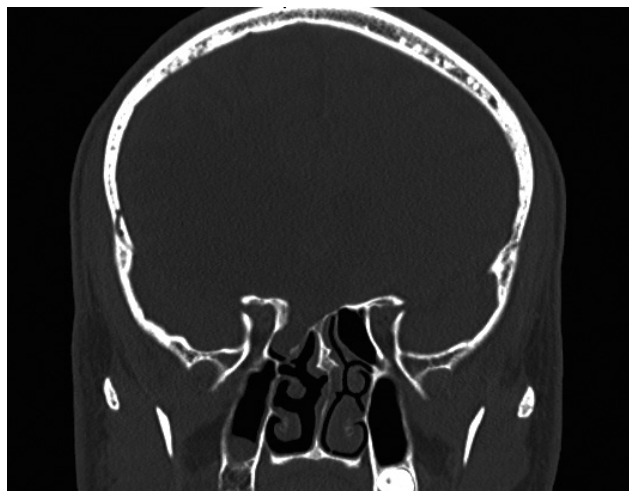
Figure 39-2. Computed tomography. Coronal orientation in bone windows demonstrating traumatic encephalocele.

Figure 39-3. Axial T2-weighted magnetic resonance imaging showing T2 hyperintense signal involving the right frontal lobe. Note multiple loculations suggestive of a mucocele.

Operative Plan

Radiographic findings led to further discussion with the soldier, who recalled being involved in an automobile collision as an adolescent. Taken together with the patient's history, the mass was now felt to represent a posttraumatic intracranial mucocele with an associated encephalocele and a probable CSF leak. Consultation with neurosurgery was undertaken to determine the approach to decompression and repair. A decision was made to approach the mucocele and skull base defect via bifrontal craniotomy with obliteration of the frontal sinus.
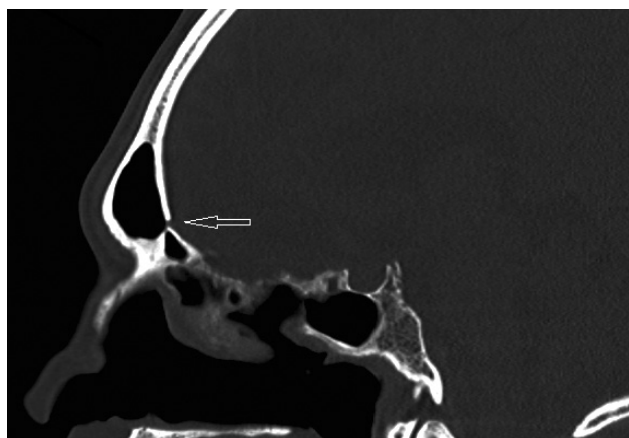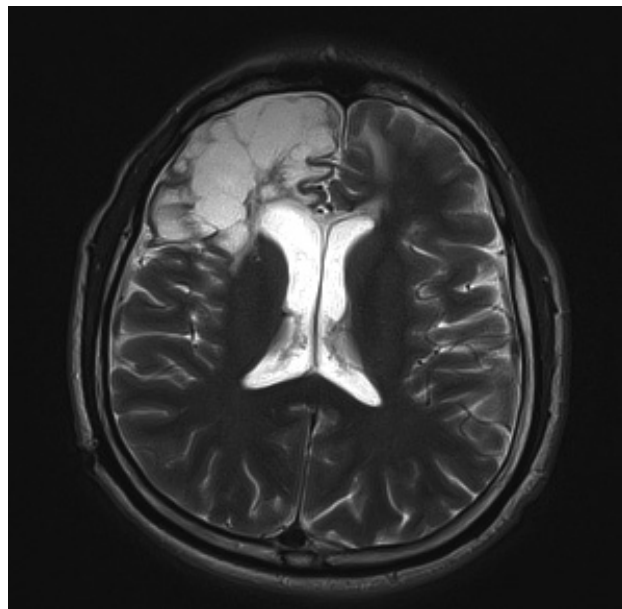
Operation

The patient underwent neurosurgery for evacuation of the mucocele. A bifrontal craniotomy was performed, with a large mucocele found extending over the optic chiasm. The mucocele was decompressed, and the skull base defect was repaired with harvested calvarium. The right frontal sinus was obliterated. On postoperative day 2, the patient
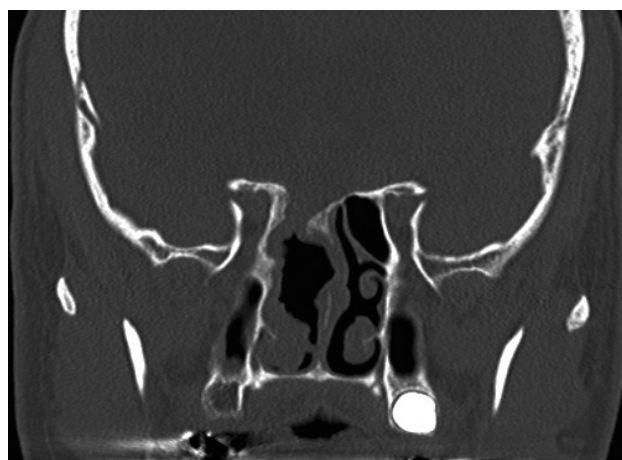

Figure 39-4. Sagittal orientation. Note dehiscence of posterior table of the frontal sinus (*arrow*).

Figure 39-5. Coronal orientation at same level as Figure 39-2, six months after encephalocele repair with pedicled nasoseptal flap.

underwent emergent craniotomy for an epidural hematoma. Nasal endoscopy was performed at the time of the second surgery, with no evidence of ongoing CSF fluid leak. Three weeks following surgery, the patient presented to the emergency department with intermittent, followed by persistent, clear rhinorrhea. Imaging after the craniotomy showed persistence of the posterior encephalocele. A lumbar drain and conservative treatment for CSF leak were unsuccessful. The patient was then taken back to the operating room and an otolaryngologist repaired the encephalocele with a unilateral pedicled nasoseptal flap. The patient reported resolution of clear rhinorrhea after the procedure. Imaging obtained 6 months after endonasal repair demonstrated no recurrence (Figure 39-5).

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